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Troublesome Reflection: Racism as the Blind Spot in the Scientific Critique of Race

Charles C. Roseman

A Troublesome Inheritance: Genes, Race and Human History, by Nicholas Wade. New York: Penguin Press, 2013. x + 278 pp. 978-1-5942-0446-3 (hardcover). US $27.95.

In *A Troublesome Inheritance*, Nicholas Wade seeks to use advances in genomics and comparative human biology to revivify hereditarian racialist notions about the ways in which differences among human societies are shaped by evolutionary forces acting on genetic variation. The arc of the argument consists of three claims: (1) human evolution has produced some unspecified number of races; (2) differences among these races in social dispositions have a strong genetic component; and (3) an oppressive academic environment keeps this kind of research out of the intellectual mainstream. These claims form the core of a world view that I refer to as "hereditarian racialism." Recent examples of this genera include *The Bell Curve* (Herrnstein and Murray 1994) and *Race, Evolution, and Behavior* (Rushton 1995), with examples extending back for some time (e.g., Grant 1970 [1912]). None of these claims are true. The book is neither good popular science writing nor all that new or interesting by the standards of the hereditarian racialist literature. (Rushton [1995] and Miele and Sarich [2005] are far more interesting examples of this genera.) *Troublesome Inheritance*, however, is a useful foil for a critical examination of the mainline scientific critique of hereditarian racialism, which, as it stands, is weak and scattered. To recuperate a useful scientific critique of race, we need to come

to grips with ways in which the political processes of racism have shaped human organisms over the last few hundred years.

Genomic Variation and Human Population History and Structure

Contrary to Wade's assertion, all parties to controversies surrounding human variation agree that humans show genomic and phenotypic variation that is structured in geographic space, through time, and across many social divisions. The disagreement is over how best to describe and model the evolutionary causes of this variation. Ignoring for the moment the large changes in the distribution of human genetic variation over the last few centuries (more on that below under Evolutionary Consequences of Racism), we can build a range of evolutionary models and statistically compare their fit to available genomic data.

In this idiom, racial models of variation like the one advocated in *Troublesome Inheritance* take the form of something like the tree diagram in Figure 1A (Hunley et al. 2009; Long and Kittles 2003). Groups within a race share common ancestry with one another more recently than they do with groups in other races. In contrast, most narrative accounts of recent human evolution feature population fissioning and founding events reflecting the movement of groups into different regions (previously occupied or not; see, e.g., Henn et al. 2012). Out-of-Africa dispersals and the spread of agriculture are two examples of these kinds of events. In this case, we might start with an estimate of a tree of patterns of common ancestry among groups under the expectation that groups with more recent common ancestry should be more genetically similar than those with distant common ancestry (Long and Kittles 2003, 2009**;** Long et al. 2009; Pickrell and Pritchard 2012). We can then add in admixture (episodic mixing of previously isolated groups) and gene flow between groups to produce a more elaborate model (Hunley et al.

2009; Pickrell and Pritchard 2012; Prüfer et al. 2014). The result is something like the elaborate model sketched out in Figure 1B, which is far and away the best fit to population genomic data. The relative importance of the different processes that make up these models are still a matter for further research, but it is certain that the racial model is a poor fit to the data and does not allow us to generate new and interesting questions.

Human Phenotypic Evolution

In the case of Wade's second claim about among-group differences in innate social propensities, he admits that much of his work is speculative, and I shall not engage with the bulk of it. He does make statements of what he regards as matters of fact about relationships between race and skull morphology. I focus on skull morphology because we know quite a bit about its evolution, and how it is used in *Troublesome Inheritance* will be an indicator of the rigor with which issues in phenotypic evolution are represented throughout the book.

Wade makes the claim that "human skulls fall into three distinctive shapes, which reflect their owner's degree of ancestry in the three main races, Caucasian, East Asian, and African" and that skulls can be matched with race with better than 80% success. Wade claims that a five-race taxonomy enjoys genomic support, and even though five does not equal three, he still gestures back to skull morphology as proof of race. The first thing to point out is that these techniques (discriminant function analysis) require races to be defined ahead of time, and then a statistical model is built that maximizes the differences between the predefined groups. The techniques do not find groups; they simply assign individuals to groups. There is not a stitch of evolutionary theory informing this practice.

If we use the discriminant function methods to test whether pairs of predefined groups are races, we end up with races galore, not the inconsistently enumerated few from *Troublesome Inheritance*. People from northern and southern Japan would come from different races, as would those from Austria and Norway (Ousley et al. 2009). Moreover, these techniques behave badly when challenged with skulls of individuals from groups not included in the samples used to build the methods and are often no better than random chance (Konigsberg et al. 2009). You will not read in *Troublesome Inheritance* that we know that random genetic drift, mutation, and gene flow have led to a pattern and magnitude of among-group cranial variation that bears a hazy resemblance to what we gather from population genomic analysis (Betti et al. 2010; von Cramon-Taubadel 2014; Lynch 1990; Relethford 1994; Roseman 2004; Weaver et al. 2008). Exceptions to this general trend appear in the form of natural selection and nongenetic effects acting in population- and region-specific ways. The rapidity of human cranial evolution is unusual among mammals and presents a vital window into issues of constraint and evolvability that are at the core of current problems in evolutionary theory (Lynch 1990; Martinez-Abadias et al. 2012; Weaver et al. 2007, 2008).

It is telling that *Troublesome Inheritance* omits any reference to this work, especially since it is a nexus between the study of phenotypes and genomes of the kind that Wade thinks brings so much explanatory power to problems in human evolution (Roseman and Weaver 2007). Instead, he bases his arguments about the cranium on a forensic literature that has no evolutionary content and offers no causal explanation for variation. The lack of rigor in dealing with this relatively simple issue makes me doubt the veracity of his claims about other aspects of phenotype.

A Badly Run Conspiracy

I think that the primary reason for ignoring the hereditarian racialist literature on intelligence and other psychological characteristics has little to do with peer pressure. This point is hard to prove and certainly merits some investigation by a qualified social scientist, but my own impression from talking to colleagues is that the avoidance stems from the fact that these traits are difficult to study, and the work that is being done on them is seen as being largely of bad quality. Contemporary scholars of human variation do not avoid talking about the interactions between biological evolution and human societies in cases where we have the data, methods, and theory to do so with rigor. The ways in which humans, plants, animals, and pathogens have coevolved during the development of agriculture and pastoralism are exciting and vibrant fields of research (Kwiatkowski 2005; Perry et al. 2007; Gignoux et al. 2011; Skoglund et al. 2014). Socially mediated genotype-by-environment interactions are also the object of increasing scrutiny (Gravlee et al. 2009).

The diversity of opinion about the causes and patterning of human variation within the scientific community (see Edgar and Hunley 2009) would seem to indicate that if there is a conspiracy to silence hereditarian racialist views, it is very badly run. Some of this variation in opinion is the result of the practice of good science, in which multiple explanations for problems are put forth and argued about. Some of the variation in opinion, however, could be a product of the fragmentation of the study of human biology (Auerbach and Cabana 2013). Molecular and morphological specialists were famously at odds with each other for years (Lubenow 1992: 83), and even specialists in different parts of anatomy do not communicate in a way that would allow for a unified view of the human organism. There is a very real risk that this conceptual drawing

and quartering of the human organism and the inconsistencies that stem from it contribute to the evolutionary sciences' less than coherent critique of hereditarian racialism.

Troublesome Reflection

For all its failures, *Troublesome Inheritance* does provide scholars of human biology with a foil that we can use to evaluate our standard critique of hereditarian racialism. My own view of the image cast back at me from Wade's book is that the critique is both weak and in need of considerable revision.

Take the oft-cited observation that the proportion of variation within races is a small fraction of the overall variation both in absolute terms and relative to other organisms (Lewontin [1972] gives the first estimate, and it is cited in places as far afield as political philosophy [e.g., Appiah 1985]). Estimating this proportion assumes that races are actually there. Without a racial or other unrealistic model, this statistic has no evolutionary interpretation (Long and Kittles 2003). The same applies to partitioning among-population variation without nesting populations into races. By deploying this fact as though it carries substantial evolutionary meaning, a critic of hereditarian racialism is arguing about how much of the variation race might account for and what social implications it might entail while implicitly conceding that a racial taxonomy is legitimate.

Likewise, scientists often appeal to isolation by distance, which proposes an equilibrium between the among-group diversifying effect of random genetic drift and homogenizing effects of gene flow among neighboring groups (Wright 1943), when talking about human variation (Eisenberg and Hayes 2011; Handley et al. 2007). This includes recent responses to *Troublesome Inheritance* in online popular science venues (Fuentes 2014; Raff 2014). Isolation by distance

fares little better than the race model when fit to human population genomic data (Hunley et al. 2009), dulling any critique of hereditarian racialism that uses it.

The most perplexing thing about both Wade's insistence that there are races in the taxonomic sense and the fixation on dispelling notions of biological race on the part of critics of hereditarian racialism is that the existence of biological races is not a necessary condition for an argument about the coevolution of society and behavior in different parts of the world. Evolved among-group differences require only genetic variation and the action of evolutionary forces (Lewontin 1975). It does not matter if spatial differences in the bulk of the genome are racially patterned or entirely clinal. Wade could have caused the critique of hereditarianism racialism a considerable bother had he pointed this out.

One point of agreement among nearly all parties is that we can say with some certainty what human variation *was* like at a single point in time and that this point in time is uniquely relevant to problems of race today. This anthropological genetic present is usually conceived of as being sometime in the mid-15th century before transoceanic European conquest and colonialism (Marks 1995). It tends to be marked through the use of the present tense. Hereditarian racialism holds that there *really are* races (read: were in the anthropological genetic present) while conceding some blurriness of boundaries between races. Its critics often claim that human variation *really is* clinal but allow that the rate of change of a trait in space can vary. Neither claim reflects the present distribution of genetic variation in social or geographic spaces, and neither question is framed in such a way that answers to them could be informative about race and genetics today.

The Evolutionary Consequences of Racism

The peril of situating arguments about genetics and race in the anthropological genetic present is that it allows us to talk about race without talking about racism. This stymies our attempts to make sense of race and genetics because the only coherent theories of race depend on the explanatory power of racism. The sociologist and philosopher W. E. B. Du Bois and his inheritors model race and racism as being coconstituted in a political process acting on multigenerational time scales (Du Bois 1903, 1920; Carbonella and Kasmir 2008; Harrison 1992; Visweswaran 1998). This is in opposition to the view anthropologists receive from Franz Boas that race is primary and that racism arises from conflict between races and can be mitigated primarily through race mixture (Boas 1921).

In this view, races—or, more properly, racialized groups—are produced as people enact political control or have political control enacted on them in the form of economic exploitation, segregation, genocide, dispossession, or other depredations. By this rationale, an African American race emerges over several generations from a disparate set of groups of people with no previous sense of kinship through the sharing of a history of slavery and struggle against it and other forms of racism (Du Bois 2007; Visweswaran 1998). Different ways of reckoning race in different parts of the world are the product of the several manifestations of racism. For the remainder of this essay I use *race* to refer to the general processes of marking individuals and grouping them through racism and "racialized groups" to refer to the groups that are formed by these processes. The dynamics of racialization as motivated by racism are historically contingent, and multiple racial formations each with their own way of delineating racialized groups have been produced at different times and places by various kinds of racism.

Coupled with an eye toward how racism governs reproduction and inheritance (Fields 1990; Hollinger 2003), this way of modeling race might clarify our view of the relationship

between race and genetics in the present day. This is particularly relevant to the role that race plays in issues in medicine and public health. In these cases, we are not so interested in the genetic variation in the Late Middle Ages as we are in the relationships among genetic variation, race, and health in the present day. This requires an explanation of how the intergenerational effects of the political management of reproduction and inheritance of status have shaped genetic variation.

By way of example, take a city in the US Midwest with a majority white population and a substantial minority representation of African Americans as tallied by the US Census. Were we to sample the genomes of the inhabitants of this city, we would find a substantial departure from Hardy-Weinberg-Castle equilibrium (HWCE), the model of what happens to genetic variation if random mating prevails and no evolutionary processes are at work. HWCE serves as the starting point for most evolutionary genetic problems, and such a large deviation from its expectation begs for an evolutionary explanation.

Genetics is the study of resemblances among relatives. Many of the ways in which race was codified into law and incorporated into custom were to govern who was to marry whom and how one could issue political status to one's offspring (Hollinger 2003; Pascoe 1996). In the case of the black/white racial dichotomy that has so defined American life, antimiscegenation statutes punished couplings across racialized groups, and rules of hypodescent ensured fidelity in the inheritance of status (Pascoe 1996). The departure from HWCE in our example is the product of racism's (and sexism's) past and ongoing roles in governing reproduction.

Thus, the allele frequency differences that we see between racialized groups as people understand and experience them today are in part the product of racism acting to shape genetic variation. So too are the vast changes in the distribution of human genetic variation seen over the

last few centuries arising from the dispossession and genocide of indigenous peoples, forced migration of enslaved people, and the establishment of colonial and settler populations. The different racisms that shaped and continue to shape racialized groups across the world mean that any racism might shape genetic variation in a variety of ways through regulation of reproduction and inheritance of status. Put bluntly, racism has been a potent driver of human evolution over the last few centuries because of the way it affects relatedness and the inheritance of genes.

Contrary to Wade's version of history, genes probably played a largely passive role in race and human affairs over the last several hundred years. Rather than natural selection evolving genetic propensities for different political systems around the world, alleles drafted along in the wake of the racisms that define racialized groups and continue to shape and govern their boundaries through the regulation of reproduction. This builds incidental correlations between racialized groups and genetic variation. Some of this genetic variation may contribute to phenotypic differences, some of which may present fairly sharp distinctions along racial lines such that they become racialized and used as a means of identification and drawing social distinctions. Race and racism also construct environments in which humans grow, live, and die, leading to a confounding of genetic and environmental effects on phenotype (Cavalli-Sforza and Feldman 1973; Gravlee et al. 2009; Gravlee 2009; Lewontin 1975; Reverby 2010). Both are inherited through different kinds of paths (i.e., genetic vs. social/legal), but the intergenerational persistence of racism can cause these modes of inheritance to covary with one another, calling into question our ability to assign the causes of difference to either environment or genes.

We cannot explain the relationships among genes, race, and health outcomes without talking about the impact of racism both on living people and on their ancestors. Everything from life span (Edwards and Tuljapurkar 2005) to sexually transmitted infections (Handcock and

Jones 2006) is affected by this historically contingent set of racial dynamics. This is for the simple reason that the present configuration of races and distribution of genetic variation is what is relevant for human well-being now and in the future. Understanding the prehistoric predicates for this variation is important to establish some sense of initial conditions, but it is insufficient for understanding the here and now. Without incorporating the effects of racism into models of human variation today, we will not be able to have a cohesive theory of genes and race, and the scientific critique of race will continue to have no teeth.

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Figure 1. Models of population structure and history. Black lines indicate evolving lineages and recency of common ancestry, with time going from left to right. The numbered tips of the lineages are operationally defined groups of individuals. The length of a line segment reflects the cumulative effect of genetic drift, not time elapsed. (A) The race model as classically conceived and argued for by Wade. Group membership in races is indicated by encompassing boxes. (B) An elaborated model with a complex pattern of common ancestry, gene flow, and admixture among groups. Double arrows indicate recurring gene flow between groups, and single arrows indicate episodes of admixture.